FOCAL CORD LESIONS.

LUMBAR POLIOMYELITIS ANTERIOR SUBACUTA UNILATERALIS.

TRAUMATIC CERVICO-DORSAL POLIOMYELITIS WITHOUT VERTEBRAL FRACTURE.

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Of Cincinnati.

Case I. Lumbar Poliomyelitis Anterior Subacuta Unilateralis in a Man of Forty-four.

F. H. J., American, of German ancestry, aet. 44, is a salesman and married. He was admitted to the Cincinnati Hospital, neurological service, March 4th, 1896.

Complaint:—Loss of power in the left leg and foot, of five weeks' duration.

Family history is good; parents are living and healthy; no nervous disease is known. One daughter of the patient has "rheumatism." (This, on examination, appeared to be an angioneurosis due to lithemia.)

Personal History:—He has had most of the children's diseases in early life, except diphtheria. Had gonorrhea in youth. He denies syphilis, and presents no evidence of the same. Has been in good social and financial circumstances until recently, when he had much domestic and pecuniary trouble. He uses alcohol moderately.

The onset of the present complaint was gradual. Five weeks ago he first noted numbness and coldness in the left toes which was followed in a few days by weakness of the ankle and by foot-drop. He has had occasional cramps in the left sole and calf. There has been no tenderness to pressure, no nausea nor vomiting. The appetite has been good.

Present State:—Physique is good; height is 5 ft. 9 in.; weight is 160 lbs. He is of dark complexion, has gray eyes and hair and looks well preserved. The heart, lungs and urine are normal. The radial arteries are elastic.

Left foot-drop is present. He walks with "high-action" step on the left side. The mental state and intelligence are good. There is no speech defect.

Cranial nerves:—Vision is good for ordinary reading. Visual fields are concentrically contracted. The pupils respond normally to light, accommodation and convergence. Smell, taste and hearing are good. There is no facial palsy nor anesthesia.

Trunk and Extremities:—Motor: muscular system generally is well developed and nourished . There are flaccid paralysis and moderate wasting of muscles of left leg and foot, both front and back. The power in the peroneal group is *nil.* He cannot flex ankle dorsally, but can flex the toes feebly. Grasp of right hand registers 80; of left 72.

Electrical Tests:—(See Chart I.) To faradism there is no response in the left peronei and extensors of the toes; sluggish response in the left tibialis anticus and calf muscles. To galvanism there is partial R. D. in all the muscles of the left leg; the contractions are very sluggish. KC > AC. Difference in poles is very slight. The muscles of the right leg and both arms responded normally to both currents.

Sensory: Tact and pain senses are normal everywhere. There is paresthesia to heat and cold over both legs, as per Chart II. He calls cold " hot " usually; hot " cold " rarely.

There is some defect of muscular sense in the left leg, foot and toes.

Reflexes:—Organic: There are no bladder nor rectal defects.

Myotatic: Knee jerks are active. L.> R. Ankle clonus is absent.

Skin: Gluteal reflex on the right side is present; on the left is absent. Epigastric, abdominal, cremasteric, plantar reflexes are absent on both sides.

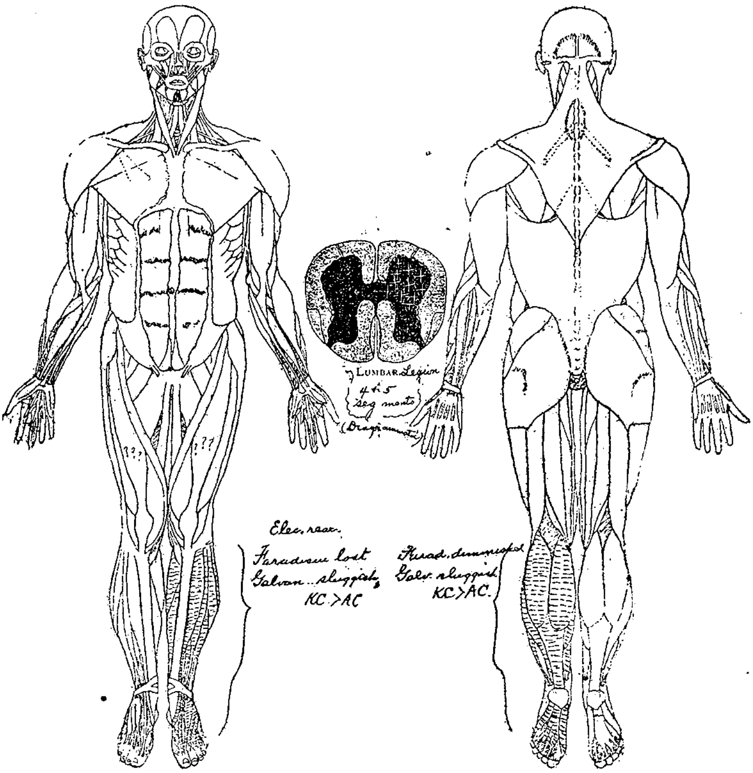

CHART I.

Lumbar Poliomyelitis Anterior Subacuta Unilateralis.
== Paralysis, rapid wasting and R. D.

There is vaso-paresis over a small area (3 × 2 inches) in the left leg (see Chart II). This appeared as a dark purplish blotch, which later faded out, leaving a pigmented area.

Trophic: There are muscular wasting and R. D., as per Chart I.

Treatment consisted of massage, passive motion, galvanism, strychnia.

March 16th.—The patient can extend left toes sligthly; paresthesia is unchanged. March 24th.—Area of thermo-

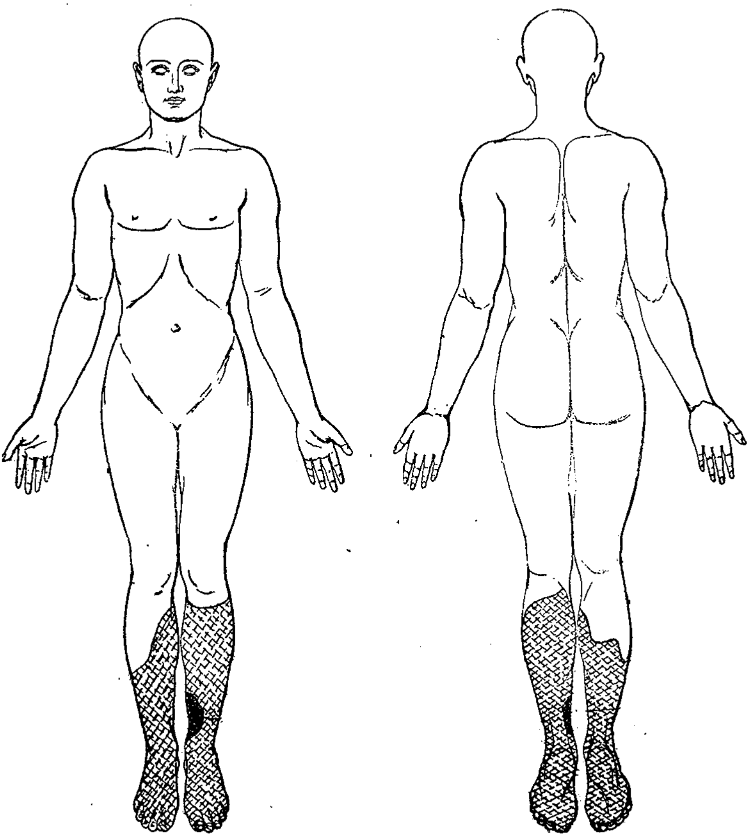

CHART II.

Lumbar Poliomyelitis Anterior Subacuta Unilateralis.

×× Area of Thermoparesthesia, March 4th, 1896. Calls cold "hot" usually; calls hot "cold" rarely. Tact and pain senses normal.

The dark-shaded oval area was purplish-red in color, and would probably have been the seat of a bedsore if subjected to pressure.

paresthesia is smaller; see Chart III. Knee jerks are normal and equal. April 13th.—Is discharged at his own request, much improved; with good use of both legs and feet. Sensation is normal. August 13th.—I saw him at my office to-day. He is practically well, and has no evidence of weakness in the left ankle to ordinary tests. No atrophy is evident. Electrical tests were not made. May

10th, 1897.—I met the patient on the street. He had no defect of gait and says he has perfect use of limb.

Comment:—This case is classified, in accordance with common custom, as a focal poliomyelitis anterior. There is every reason to believe, however, that the primary lesion was a thrombosis, to which the inflammatory process was secondary. Williamson,[2] and others, have adduced strong evidence in favor of this view, which is also in accord with modern ideas of the causation of infectious diseases and softenings of the nervous system in general. Syphilis, as a cause for the thrombosis in the case here reported, would appear to be excluded by the absence of any history or objective evidence of that disease, as well as by the complete recovery without antisyphilitic treatment. The real cause, therefore, remains unknown.

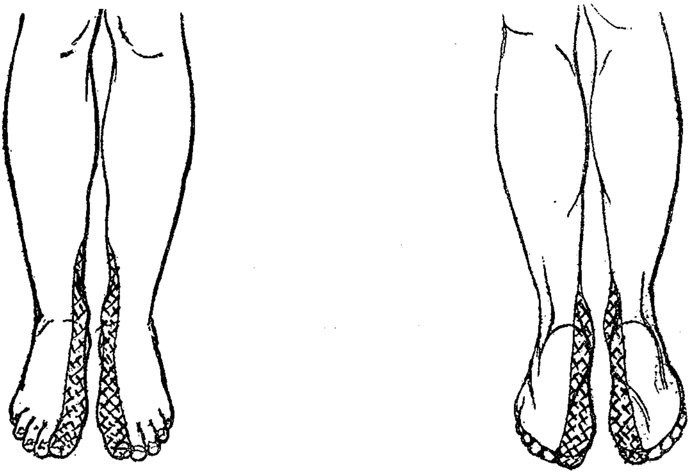

CHART III.

Lumbar Poliomyelitis Anterior Subacuta Unilateralis. Thermoparesthesia, twenty days later.

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[2] On the Relation of Diseases of the Spinal Cord to the Distribution and Lesions of the Spinal Bloodvessels. By R. T. Williamson, M.D., (Lond.) M.R.C.P., 1895. (Reprint with additions from the Manchester Medical Chronicle, 1894 and 1895.)

Case II. Traumatic Cervico-dorsal Poliomyelitis Without Vertebral Fracture.

H. P., colored, aet. 49, is a laborer. He was admitted to the Cincinnati Hospital, Sep. 25th, 1896, and was assigned to the surgical service of my colleague, Dr. N. P. Dandridge.

He was unconscious on arrival but became conscious twenty minutes later and gave a history of having fallen thirty feet into a cellar.

He complained of pain in the back and left shoulder. The shoulder was swollen, abraded and tender. A small scalp wound was seen on the vertex. There were no signs of injury to the vertebræ and no paralysis. Temperature upon arrival was 103.4 deg. Treatment consisted of an ice-cap to the shoulder.

Oct. 1st.—The swelling in the shoulder is about gone. Temperature is normal and he walks about the ward.

Oct. 3d.—Power is lost in the fingers of both hands.

Oct. 12th.—He was transferred to the neurological service.

Neurological examination, Oct. 12th, 1897, is as follows: He is a dark negro of average height, strong build and excellent muscular development. Pulse 64, regular, and there is no heart lesion. Urine is 1023, alkaline; contains no albumin, nor sugar, nor sediment.

His mental state is good, and he has no speech defect.

Cranial nerves: Vision is good to rough tests and there is no nystagmus. Pupils are moderately contracted; their dilatation is sluggish and extremely limited in extent. The pupils contract readily to light, accomodation and convergence. No other cranial nerve defects are noted.

Trunk and extremities—Motor: The muscles generally appear well nourished. There is no rigidity. Abduction and circumduction are present in both shoulders. Flexion at elbow is present and fairly strong in both arms. Extension is practically *nil.* Flexion and extension are very weak at the wrists; *extension* is *strongest.*

Grasp:—Dynamometer, R.O., L.O. Slight pressure of one finger at wrist easily controls triceps. The muscles of both forearms and hands are wasted and flabby. The costal portion of each pectoralis majora is also (see Chart IV). All movements of the lower extremities seem to be normal in extent and power.

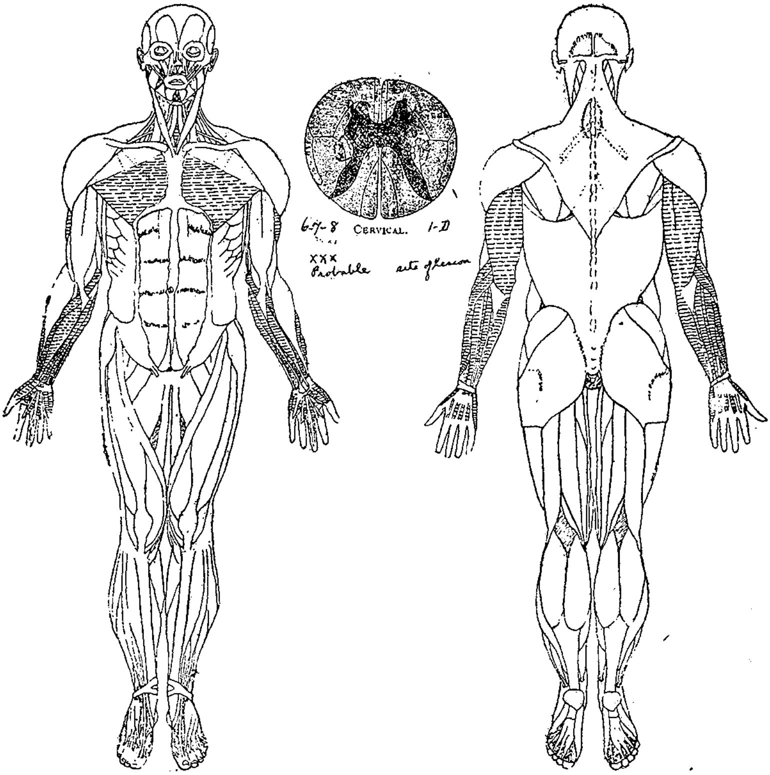

Chart IV.

Traumatic Cervico-dorsal Poliomyelitis Without Vertebral Fracture.

== Paralysis, rapid wasting and R. D. (Very sluggish response to faradism; no response to galvanism 15 M.A.)

Electrical Tests (see Chart IV.): Faradism yields a very sluggish response in the muscles shaded in the chart.

Galvanism: It is almost impossible to get any response in the same area with a bearable current (15-20 M.A.) (the skin is very dry and harsh). Response is good in the muscles not shaded.

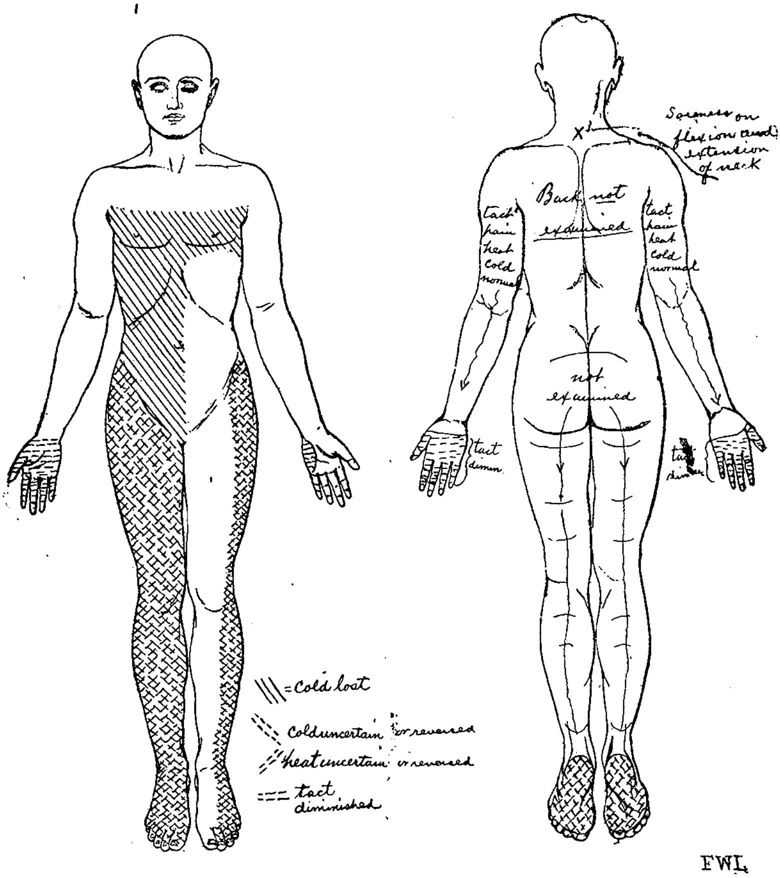
Soreness on flexion and extension of neck
X
Back not examined
tact pain heat cold normal
tact pain heat cold normal
not examined
tact dimin.
tact dimin.
\\\ = Cold lost
\\ Cold uncertain or reversed
// heat uncertain or reversed
=== tact diminished
FWL

CHART V.

Traumatic Cervico-dorsal Poliomyelitis Without Vertebral Fracture.
\\\\ Cold lost (heat preserved).
\\ Cold uncertain or reversed.
// Heat uncertain or reversed.
== Tact diminished.

Sensory: Some soreness, not excessive, on flexion and extension of the neck. Tact and pain senses are diminished over a small area on the hand (see Chart V). There are extensive defects of heat and cold senses as per Chart V. It was not thought advisable to turn the patient in order to test the back.

Reflexes—Organic: There are no defects of deglutition nor respiration, and no bladder nor rectal defects.

Myotatic: Jaw jerk is absent; elbow jerk is present and moderate on both sides. The wrist jerk is absent on both sides; knee jerks are present and active, R.>L. Ankle clonus on the right side is spurious; on the left side it is absent.

Skin: Palmar, epigastric, abdominal, cremasteric reflexes are absent on both sides. Plantar is present on both sides.

Vasomotor: Skin of forearms and hands is very dry and harsh.

Trophic: There is marked muscular wasting and R. D. as per Chart IV. No bed sores (now or later).

Diagnosis: Traumatic, focal (capillary?) lesion of 6, 7, 8, cervical and 1 dorsal segments, followed by poliomyelitis.

Treatment: Absolute rest; liquid diet; mercurial inunctions and iodide of potassium vigorously pushed.

Oct. 21st. Sensation: Heat and cold senses are uncertain (not lost) over entire area from nipple line to the feet anteriorly. Tact and pain senses are diminished on the hands, as before.

Nov. 20th. He is doing well, and has some power in the hands. The flexors and extensors of the hands respond feebly to faradism and galvanism.

Nov. 23d. He is improving rapidly; all sensations are good, and there is good faradic response in all the wasted muscles. The galvanic response is sluggish. The dilatation of the pupils in darkness is still sluggish and limited; contraction to light is active. The muscular power is improving.

Nov. 25th. He had a chill at 4 p.m., followed by a temperature of 104.6 degrees. Blood examination was negative. Quinine was given.

Nov. 26th. Temperature is normal.

Nov. 29th. Grasp: dynamometer, R. 17½, L. 15 (lower scale, numerically speaking.) Temperature varied from normal to 104 degrees, until Dec. 4th, when a fluctuating swelling was noted at left sternoclavicular joint.

Dec. 13th. He was transferred to the surgical service for operation. A purulent-looking fluid was evacuated from the left sternoclavicular region. Fever continued, and extensive mediastinal suppuration became apparent.

Jan. 1st, 1897. The patient died of pyemia. Autopsy was not obtained.

Comment: This case presents several features of interest aside from those of localization:

First.—The temperature of 103.4 degrees, a few hours after a severe injury was the only symptom referable to the spinal cord for eight days after the injury. The temperature fell to normal within a day or two, and did not rise again above normal for a month, when suppuration developed in the mediastinum. Five days after the fall the patient was up and about the ward, in his usual health and with no noticeable paralysis. Three days later (eight days after the fall) he noticed weakness in both hands, which rapidly progressed to complete paralysis, rapid wasting and R.D. of the muscles of hands, forearm, triceps and the costal pectoralis.

Secondly.—There appeared no evidence, subjective or objective, of any fracture of the vertebræ.

Thirdly.—The distribution of the lesion, as indicated by the symptoms, was limited almost entirely to the gray matter, as shown by the entire absence of bladder or rectal symtoms, as well as of leg paralysis or marked spastic symptoms. This would seem to the writer to indicate that the softer consistence of the cinerea, affording less support to its bloodvessels, had permitted the formation of multiple minute vascular lesions, which were followed by numerous capillary thrombi, insufficient in themselves to cause immediate symptoms, but capable of making their presence evident after some days by exciting multiple foci of round-cell infiltration. This exudate was sufficient to cause pressure and abolition of conducting function, with only partial destruction of neuron bodies and axons, as is indicated by preservation of sluggish faradic response and by the rapid improvement in muscular power, the rate of improvement being too rapid to be ascribed to reproduction of axons. That complete recovery would have occurred had the patient lived, is, of course, not likely, since some permanent destruction must have resulted from consecutive sclerosis.

Fourthly.—The medico-legal aspects of the case are significant. In a case recently the subject of litigation, in which the writer was called to testify, there was good reason to believe that the cervical cord was the seat of organic changes, due to injury. The question of fracture, or non-fracture, of the vertebræ appeared to be one of great importance at the trial, the inference on one side being that if there were no fracture, the cord was not structurally damaged. This view is certainly not tenable, having in view the above and similar cases. Nor does it seem at all necessary at the present day from a neurological standpoint to prove a fracture of bone in order to establish organic injury to nerve centres.

The lapse of time (eight days) before marked symptoms of spinal injury, in the case here reported, is another point likely to give rise to forensic debate as to the connection between the injury and the myelitis.

Fifthly.—While there was no history nor marked evidence of syphilis as a factor in the case, yet mercury by inunction and iodide of potassium were pushed freely, with excellent effects, as would doubtless be the case in any event with such a recent exudate.

The actual cause of death, the mediastinal suppuration, is to be looked upon probably as the result of the fall, and was aided, perhaps, by the damage to the neurotrophic areas of the cord connected with that region.

Finally, the two cases here reported, taken together, would seem to throw additional light on the respective pathways in the gray matter of temperature and pain impulses.

It is well known that, in most cases of lesion limited to the gray matter of the cord, and especially in syringomyelia, temperature and pain conduction are affected together; tact escaping. In both the present cases, however, the temperature sense was notably impaired or lost, while the pain and tact senses were not appreciably affected. This would seem to indicate, not only separate pathways for temperature and pain, but also *considerable distance between them;* the temperature tract, for instance, would seem to cross to the opposite side in the anterior gray commissure, while the pain pathway would seem, as suggested by Turner[3], to cross more posteriorly and to pass upward in the *substantia gelatinosa rolandi* of the posterior horns. This region apparently escaped damage in both cases here reported. Turner and Mackintosh, however, in a later paper,[4] deny the necessity of assuming the existence of separate paths in the cord for the different sensations, considering that the dissociation of the temperature and pain senses is explicable by the hypothesis that "the more fundamental the form of sensation, the more extensive is the provision for its conduction in the cord." Hence, according to their view, temperature sense (vibration?), a late acquirement, is lost first, pain next, and touch last; the return in cases of recovery being in inverse order.

It would seem to the writer, however, that separate neuron-complexes for the conversion or elaboration of pain and temperature impulses may readily exist *in the*

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[3] The Results of Experimental Destruction of the Tubercle of Rolando. By W. Aldren Turner, M.D., F.R.C.P. Brain, 1895, p. 231.

[4] Three Cases of New Growth with Cavity Formation in the Spinal Cord. By W. Aldren Turner, M.D., F.R.C.P., and Ashley W. Mackintosh, M.D. Brain, 1896, p. 331.

cord, without the necessity for separate *peripheral* pain and temperature neurons. In other words, amongst the numerous short neuron tracts of the cord the spinal function may exist of converting (separating) heat and pain vibrations of the peripheral neuron into temperature and pain conduction respectively. It would seem to be rendered probable, by the two cases here reported, that this separation occurs in the cord; the lesions in the anterior horns and central gray matter accounting for the temperature loss; while the escape of the substantia gelatinosa rolandi and of the columns of Goll and Burdach explains the persistence of pain and tact senses.

My acknowledgments are due to Drs. G. C. Schaeffer and D. E. Robinson, the House Surgeons, for their careful notes on these cases; and to my colleague, Dr. H. H. Hoppe, for his courtesy in permitting use of later notes made during his term of service.